Uncontrolled ventricular rate in atrial fibrillation
A manifestation of dissimilar atrial rhythms[1]

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SUMMARY A patient with coarse atrial fibrillation and a rapid ventricular response developed periods of high grade atrioventricular block interspersed with periods of rapid ventricular conduction after the administration of digitalis and propranolol. Intracardiac atrial recordings showed dissimilar atrial rhythms of high right atrial flutter and left atrial fibrillation. The low right atrial recordings showed flutter during the periods of fast ventricular rates and fibrillation during periods of slower ventricular rates.

Atrial fibrillation is a rhythm in which the ventricular response is usually well controlled at the atrioventricular node level with digitalis or propranolol. Inability to control the ventricular rate is not an uncommon problem and is usually explained on the basis of inadequate digitalis or propranolol dosage, an increase of the sympathetic tone (or decreased parasympathetic tone) on the atrioventricular node, an inordinately short atrioventricular node refractory period, or an accessory atrioventricular conduction pathway (Schamroth, 1971). We report a patient who presented with atrial fibrillation and a poorly controlled ventricular response. The unique findings of her electrophysiological study are the basis of this report.

Case report

A 33-year-old woman presented with increasing shortness of breath and a rapid irregular pulse. At age 8 she experienced an episode of acute rheumatic fever. Because of dyspnoea on exertion and easy fatigability, cardiac catheterisation was performed in 1969 when she was 22 years old, and moderate mitral regurgitation, moderate mitral stenosis, and trivial aortic regurgitation were demonstrated. Her mitral valve was replaced with a Starr-Edwards prosthesis, and her clinical condition improved. Over the subsequent 7 years, she reverted from sinus rhythm to atrial fibrillation three times, despite the chronic administration of antiarrhythmic agents. Each time she was successfully cardioverted

back to sinus rhythm. The recent development of a rapid irregular pulse and progressively worsening shortness of breath began one week before admission. Her treatment consisted of digoxin, 0·25 mg orally daily, and warfarin sodium.

The physical examination at admission disclosed a praecordial auscultatory heart rate of 170/minute and an irregular radial pulse of 110/minute. Supine right arm blood pressure was 190/60 mmHg. No discernible jugular venous pulsations were noted. The intensity of the prosthetic opening and closing sounds was diminished because of the tachycardia. A grade 2/6 crescendo-decrescendo blowing systolic murmur was present along the left sternal border and at slower rates, a grade 2/6 decrescendo aortic regurgitation murmur was present in the same location.

The chest x-ray film (posteroanterior and left lateral projections) showed mild cardiomegaly, left atrial enlargement, and pulmonary vascular redistribution. The echocardiogram showed normal prosthetic valve motion, with a poppet excursion of 1 cm and an S2-opening click interval of 90 to 110 ms. Coarse atrial fibrillation was noted during transient slowing of the heart rate on the scalar electrocardiogram.

The hospital course was characterised by considerable difficulty in bringing the ventricular rate into an acceptable range. A total of 0·375 mg digoxin was administered orally over the first 12 hours, attaining a serum level of 1·3 ng/ml (1·66 nmol/l) (normal range: 0·8 to 2·4 ng/ml (1·02 to 3·07 nmol/l)). Between 12 and 24 hours after admission the rhythm continued as coarse atrial fibrillation; however, the ventricular response
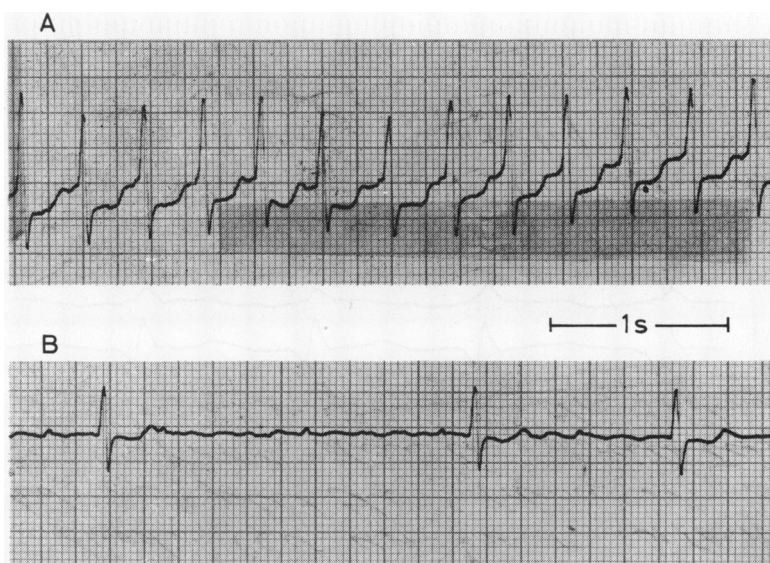
Fig. 1 *Sample monitor electrocardiographic tracings obtained after the administration of digoxin. The ventricular response of A is rapid (rate: 160-180/minute) and somewhat regular. Fifteen seconds later, the ventricular response decreased dramatically and became irregular as noted in recording B.*

underwent wide fluctuations ranging from 180/minute down to 30/minute (Fig. 1). During the periods of fast ventricular response, the patient developed dyspnoea and near syncope. Increasing the digitalis dosage (or administering propranolol) episodically decreased the ventricular response to less than 30/minute, and decreasing the dosage resulted in sustained periods of tachycardia (160 to 170/minute). This therapeutic dilemma prompted the performance of an electrophysiological study in order to determine if atrial events were in part responsible for the wide variation of ventricular rates and for the inability to decrease the ventricular rate without evoking periods of distinct bradycardia.

ELECTROPHYSIOLOGICAL STUDY AND RESULTS

Two bipolar electrode recording catheters were introduced into the right antecubital vein, and were placed in the right atrium under fluoroscopy. One catheter was positioned in the high right atrium and the other in the medial aspect of the low right atrium, adjacent to the tricuspid valve. These catheters provided electrograms of the high right atrium and the low right atrium. An oesophageal bipolar electrode catheter was positioned at the level of the mid-left atrium for recording left atrial electrograms (Barold, 1972). The signals were amplified and recorded in a frequency range of 30 to 500 cycles/second by an Electronics for Medcine DR-12. Scalar electrocardiographic leads I, aVF, and praecordial V1 were recorded simultaneously with the atrial electrograms.

The left atrial electrogram showed rapid irregular potentials, consistent with fibrillation, throughout the study. The simultaneous high right atrial recordings showed the presence of rapid regular deflections (rate 335 to 340/minute) characteristic of atrial flutter. The low right atrial recordings were of particular interest in that the rhythm in the low right atrium fluctuated between fibrillation and flutter (Fig. 2a and b). The ventricular rate during the low atrial flutter was rapid (150 to 170/minute) and during low atrial fibrillation was considerably slower (50 to 100/minute). Attempts to pace the right atrium into persistent fibrillation were not successful.

After refusing cardioversion, three doses of quinidine sulphate (300 mg orally every 6 hours) were administered in an attempt to convert the arrhythmia to sinus rhythm. She developed salvos of ventricular tachycardia and gastrointestinal distress with the quinidine and this treatment had to be discontinued. Because of the inability to convert the flutter-fibrillation to sinus rhythm, an alternative treatment was devised. A ventricular demand pacemaker was inserted into the right ventricle and digitoxin 0·1 mg orally daily and

Fig. 2(a) *Right and left atrial electrograms taken during the rapid ventricular response. The left atrial (LA) recording shows fibrillation and both high right atrium (HRA) and low right atrium (LRA) show a regular tachysystole-atrial flutter. F = flutter waves. (b) Right and left atrial electrograms obtained during a slower ventricular rate. The high right atrium (HRA) recording shows atrial flutter and the left atrium (LA) shows fibrillation. The low right atrium (LRA) at this time shows the irregular rapid rhythm of fibrillation.*

propranolol 40 mg orally every 6 hours was administered. The digitoxin and propranolol maintained the resting heart rate at less than 100 beats per minute and the pacemaker intervened at ventricular rates less than 70/minute. During a 3-month follow-up, there was considerable improvement of the patient's dyspnoea and fatigability.

Discussion

This study shows that inadequate control of the ventricular rate during atrial fibrillation may be on the basis of dissimilar atrial rhythms. The right atrial flutter became problematic in this patient only when the flutter extended into the low right atrium—atrioventricular node region. During the periods of low right atrial flutter the ventricular rate tended to become regular and increased considerably, and during fibrillation the ventricular

response decreased and became irregular. The QRS configuration during the rapid ventricular rate was similar to that during the slow rates, suggesting that a bundle of Kent accessory pathway did not account for the rapid conduction, though an atrioventricular node bypass fibre (James fibre) cannot be excluded. However, the ability to block the atrioventricular conduction with digitalis and propranolol makes the presence of an atrioventricular node bypass fibre unlikely. The difference in the atrioventricular node transmission is best explained on the basis of concealed conduction (Langendorf *et al.*, 1965), which is greater for the higher frequency irregular bombardment of fibrillation than for slower regular impulses of flutter. The atrioventricular node conduction of the two arrhythmias was strikingly different and virtually made it impossible pharmacologically to block the rapid conduction of flutter without severely blocking

atrioventricular node conduction during the periods of low right atrial fibrillation.

Several different combinations of atrial electrical events and dissimilar rhythms may present with coarse atrial fibrillation on the scalar electrocardiogram (Zipes and DeJoseph, 1973; Leier and Schaal, 1975, 1977). It is believed that in dissimilar atrial rhythms the atrial activity of the right atrium will dictate the behaviour of the atrioventricular node and the ventricular response. The right intra-atrial dissimilar rhythms of this patient clearly showed that the rhythm in the low right atrial—atrioventricular node region determines the conduction properties of the atrioventricular node regardless of what the rhythm(s) are in the remainder of the atria. The atrial refractory period of the low right atrial region probably determined whether this area would be in flutter or fibrillation (Zipes and DeJoseph, 1973). As the refractory period shortened, the fibrillation extended from the left atrial region into the low right atrium, pushing the flutter-fibrillation interface higher in the right atrium. Reciprocally, as the low right atrial refractory period increased the flutter front moved into this region.

While there are several reasons for a rapid ventricular response during atrial fibrillation, it is apparent that dissimilar atrial rhythms with flutter or another tachysystole in the low right atrial—atrioventricular node region are another cause.

Patients with coarse atrial fibrillation or flutter-fibrillation and an uncontrolled ventricular response, should undergo atrial recordings to define the atrial rhythms present, particularly in the low right atrial—atrioventricular node region. In our patient, this procedure led to the proper therapeutic approach—pacemaker placement and pharmacological atrioventricular blockade. Had pharmacological therapy alone been pursued, there may have been a fatal outcome.

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